various north shore beaches of Oahu. Both are also known to be potent tumor promoters in two-stage carcinogenesis in mouse skin.[10] The potential of these two toxins as tumor promoters in humans is unknown; but their noxious effects are a public health concern. Swimmers and seaweed on leeward shores of the Hawaiian islands rarely come in contact with blue-green algae because trade winds usually transport the algae away from the shore. Slightly elevated enterococci levels in seawater might serve as a signal for a potential health threat of harvesting seaweed for consumption.

Environmental stresses on the seaweed possibly allowed for colonization of the blue-green algae. Previous studies have demonstrated that stress from over-harvesting might cause seaweed to produce toxins as a method of protection and that environmental changes and increased pollution promote colonization of toxin-producing bacteria.[11] Although unlikely in this case, other studies have suggested that some species of seaweed may become toxic at the end of their reproductive cycle, and therefore exhibit a seasonal variation in toxin production.[12]

Although this was the first reported case of seaweed-induced illness in Hawaii, several cases have been reported in the Pacific Rim.[3,13] In 1991, 13 people became ill eating seaweed (*Gracilaria tsudai*) harvested in Guam[1]; 3 died. In addition to gastrointestinal illness, they experienced fever, wheezing, muscle fasciculations, and hypotension. In 1992, according to a press report, 3 people became ill, one of whom was hospitalized, after eating seaweed (*Grasilariopsis lemanaeformis*) picked on the beach of Half Moon Bay in California.[2] In 1993, 2 people became ill, 1 of whom later died, after eating *Gracilaria verrucosa* seaweed in Japan.[3] In the cases in Guam and California, the implicated seaweed was not previously known to be toxic. In the two cases that occurred in Japan, the seaweed was not examined adequately and no toxin was identified. Whether the dearth of reported seaweed-associated illness reflects failure to report illness or infrequent occurrence is unclear.

Since the incident in Hawaii, physicians were alerted to the potential for seaweed-induced toxicity and were requested to report cases to the Hawaii Department of Health. Seaweed has been collected from the same harvest site for toxin surveillance. Until further information regarding seaweed toxicity is known, those who harvest seaweed for consumption should be aware that any seaweed harvested from Hawaiian waters is potentially toxic. Those who experience a burning sensation after eating a seaweed preparation should notify the Hawaii Department of Health.

Although the species of blue-green algae was not identified in this outbreak, *Lyngbya majuscula*, which possibly contains debromoaplysiatoxin and aplysiatoxin and can grow epiphytically on certain edible seaweeds,[8] has been identified in Hawaii.[9] The Hawaii Department of Health recommends that seaweed to which blue-green algae—specifically *Lyngbya* species—is attached should not be eaten. Boiling the seaweed did not inactivate the toxin in the two incidents on Maui and Hawaii. Because the consumption of seaweed is common among many ethnic groups who reside in the Pacific area, additional research is necessary to determine the etiology and extent of seaweed-associated illnesses and to characterize such illnesses.

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Sphenoid Pneumoceles Cause Episodic Pressure-Related Blindness

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THOUGH ETIOLOGIES VARY, transient visual loss in adults is most commonly caused by vascular insufficiency secondary

(Hanasono MM, Norbash AM, Shepard K, Terris DJ. Sphenoid pneumoceles cause episodic pressure-related blindness. West J Med 1998; 169:295–299)

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to atherosclerotic microemboli or vasospasm of ophthalmic supplying arteries. Rarely, transient visual loss has been associated with disease of the sphenoid sinus, which is adjacent to the optic nerve.[1,2,3] Such cases result from increased pressure within the sphenoid sinus transmitted to the optic canal through a defect in the bony sinus wall. The following case report presents an unusual case of a sphenoid sinus pneumocele which manifested with visual disturbances at high altitudes due to increased air pressure in the sphenoid sinus which was transmitted to the optic nerve in the absence of infection within the optic canal.

Case Report

A 35-year-old avid snow skier with a six-month history of visual disturbances in his left eye reported that on car trips to the Sierra Mountains in Lake Tahoe, California, he would notice visual dysfunction in his left eye that began precisely at an elevation of 5,000 feet, progressing from "seeing stars" and "loss of contrast" to complete left eye blindness. These episodes typically lasted 15 to 20 minutes. This same sequence of visual loss was noted during multiple plane flights, usually beginning 20 minutes after takeoff and also lasting for 15 to 20 minutes. Each episode was accompanied by a left retroorbital headache. He was seen by an otolaryngologist who ascribed the episodes to sinusitis and prescribed methylprednisolone for one day preceding travel as well as Entex (phenylpropanolamine and guaifenesin) and acetaminophen one hour prior to travel. The headaches and visual disturbances continued despite this treatment. The patient found that placing his head between his knees caused the visual disturbances to resolve faster, often within five minutes.

A comprehensive ocular exam by an ophthalmologist and a neuro-ophthalmologist was unremarkable. One month after the onset of his symptoms, while traveling by plane in Europe, the patient developed the usual visual changes accompanied by a left-sided headache and was diagnosed with a retinal hole with operculum during an examination at an eye clinic. This hole was repaired with a laser, but his visual changes and headaches continued to occur during high altitude travel. Computed tomography (CT) and magnetic resonance imaging (MRI) scans that subsequently were obtained demonstrated left sphenoid sinusitis *(see* Radiologic Findings*)*. The patient was treated with doxycycline and methylprednisolone. He was kept on azithromycin prophylactically, but this failed to prevent the visual disturbances. A neurologist was consulted who made a diagnosis of ocular migraine and prescribed verapamil prior to flying. The visual disturbances continued despite the patient taking increasing doses of verapamil. The patient was then evaluated at the Division of Otolaryngology/Head and Neck Surgery at Stanford University Medical Center. A review of both imaging studies revealed protrusion of the left optic nerve into the superolateral sphenoid sinus.

The patient's past medical history included a three-year history of left-sided headache, morning nasal congestion that was worse on the left, and recurrent nosebleed from the left nostril for many years. He used no medications other than those described above. He was a nonsmoker and reported drinking one glass of wine with dinner each night.

A physical examination showed that, though the patient's nasal septum was deviated to the left, he had no other remarkable physical findings relevant to the head and neck. The patient had normal ocular exam including full visual fields and normal visual acuity.

A translabial, transseptal sphenoidotomy and septoplasty were performed with wide enlargement of the sphenoid ostium and removal of the sphenoidal septum. A transseptal approach was chosen over a transnasal endoscopic approach because of the greater exposure it afforded. Intraoperatively, protrusion of the left optic nerve was apparent within the superolateral wall of the left sphenoid sinus by endoscopy. There was no fluid within the sinus at the time of exploration. Mucosal tissue from the right and left sphenoid sinuses were submitted for pathologic examination. Both specimens were histologically normal. Bacterial cultures from the left sphenoid sinus mucosa grew peptostreptococci species. The patient recovered without complications and has had no further visual dysfunction or retroorbital headaches after several high altitude travel challenges. He remains free of symptoms 18 months after surgery.

Radiologic Findings

Direct coronal CT examinations of the paranasal sinuses were obtained. Coronal sequences consisted of 3 mm thick sections obtained at 4 mm intervals. The coronal CT images demonstrate soft tissue fullness in the roof of the left sphenoid sinus (Figure 1). The bony covering appears to be intact but attenuated on the 3 mm thick section using a standard soft tissue algorithm at a bone window.

MRI sequences included T1 weighted unenhanced sagittal, dual echo T2 weighted unenhanced axial, T1 weighted unenhanced and enhanced axial and coronal sequences. Additional thin-section sagittal and coronal enhanced sequences were obtained, centered on the sphenoid sinus. The MRI study demonstrates nonenhancing layering dependent fluid in the left sphenoid sinus on the enhanced T1 weighted axial sequence (Figure 2). The conventional unenhanced coronal and enhanced thin-section coronal sequences show that there is soft tissue fullness in the roof of the left sphenoid sinus that is nonenhancing, and, therefore, most likely represents the posterior layered fluid that was shown on the axial sequence and has assumed the most dependent portion of the sinus (Figure 3). Hyperintensification of the layering on T2 weighted images, corresponding with presumed fluid or mucosal thickening, was also seen. Additionally, these sequences show that there is no perceptible bony separation either from the medial inferior aspect of the left optic tract or from the medial wall of the cavernous carotid artery, thereby suggesting dehiscence.

Discussion

Pneumoceles of the sphenoid sinus in which air pressure within the sphenoid sinus compresses the optic

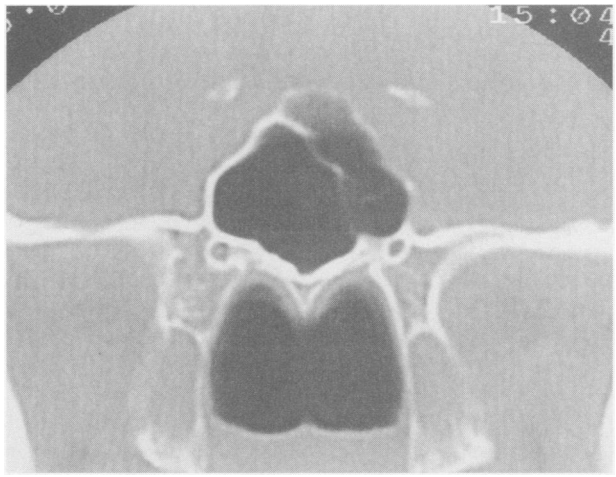

Figure 1.—Coronal standard algorithm bone window computed tomography (CT) demonstrates fullness in the roof of the left sphenoid sinus. Although the bony covering appears to be intact on this 3 mm thick section, the superolateral wall of the left sphenoid sinus was dehiscent on endoscopy.

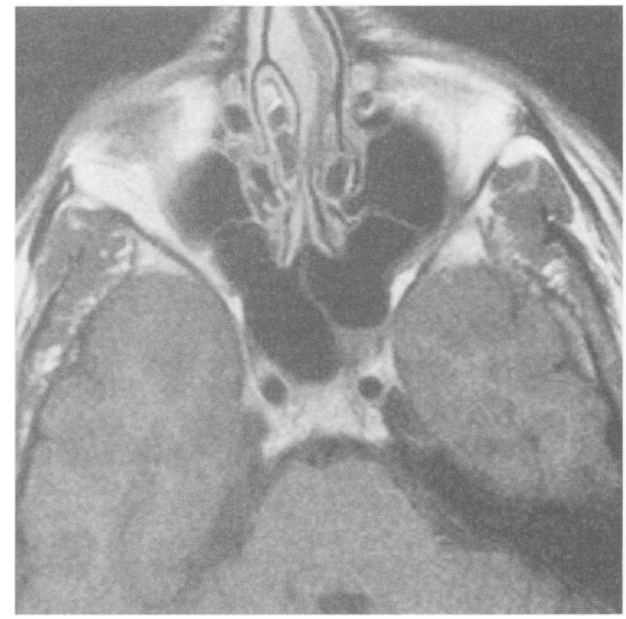

Figure 2.— Enhanced axial magnetic resonance (MR) study demonstrates nonenhancing layering fluid in the left sphenoid sinus. A concha bullosa involves the right middle turbinate. A nasal septal deformity is also seen.

nerve through a defect in the bony sinus wall causing transient visual loss have been reported on three occasions (Table 1).[1,2,3] As in the previous case, these lesions were symptomatic during ascent in aircraft. Wide sinusotomy and, in one case, the removal of a nasal polyp at the sinus ostium resulted in improvement or eradication of the symptoms.

The visual changes in the case previously presented were the result of a relative increase in pressure on an optic nerve that projected into the superolateral sphenoid sinus. The relative increase in pressure within the sinus was caused by a decrease in atmospheric pressure with ascent to higher altitudes combined with the occlusion of a poorly patent sphenoid sinus ostium preventing pressure equalization. The patient's altitude-induced visual changes were fully relieved by enlarging the sphenoid ostium, allowing decompression, drainage of the sinus, and pressure equalization.

The patient's vision changed while driving in the mountains at an altitude of approximately 5,000 feet as

TABLE 1.—*Overview of Reported Cases of Sphenoid Sinus Pneumoceles*

Report	*Age*	*Sex*	*Symptom Presentation*	*Duration*	*Findings*	*Surgery*	*Outcome*
Sugita et al (1977)	26	M	Bilateral blindness	7 y	Enlarged sinus, wall defects	Transmaxillary sphenoidotomy	No further episodes; improved vision
Som et al (1983)	48	M	Left blurred vision to blindness	2 y	Enlarged sinus, very thin or dehiscent sinus wall	Transnasal sphenoidotomy	Cure
Bell et al (1995)	41	M	Left visual field loss to blindness	1 y	Enlarged left sinus, suspected wall defects	Endoscopic ethmoidotomy, sphenoidotomy	Cure
Hanasono et al (1998)	35	M	Left visual disturbance to blindness	1 y	Enlarged left sinus, suspected wall defects	Transseptal sphenoidotomy, septoplasty	Cure

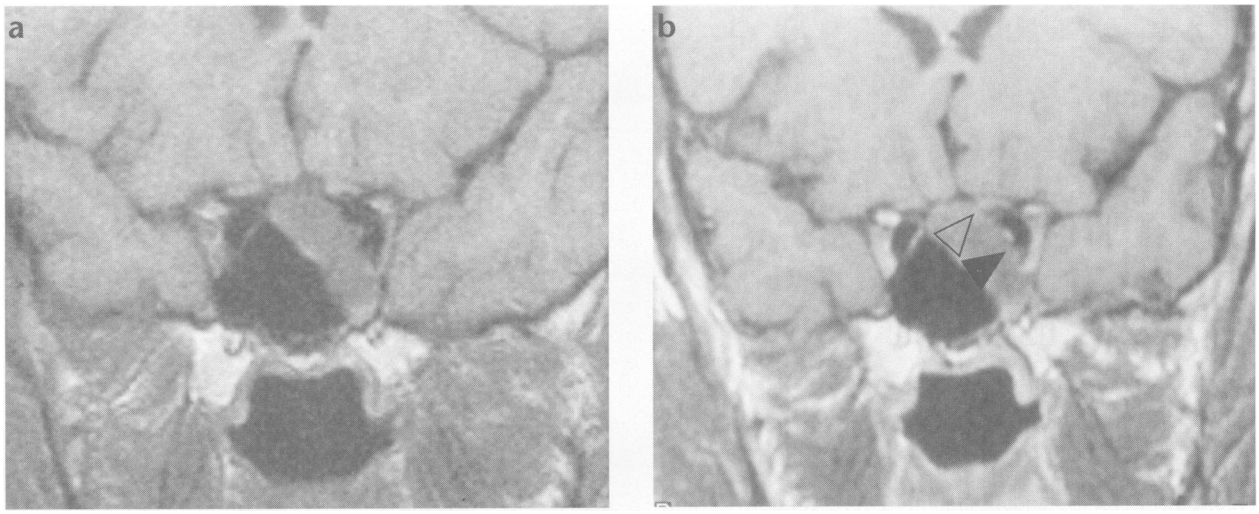

Figure 3.—The conventional unenhanced coronal (a) and enhanced thin-section coronal (b) images show nonenhancing soft tissue fullness in the roof of the left sphenoid sinus. There is no perceptible bony separation either from the medial inferior aspect of the left optic tract (△), or from the medial cavernous carotid artery (▲). There is no significant contrast enhancement of the abnormal sphenoid opacity.

well as during commercial air travel about 20 minutes after takeoff. Most commercial airplanes fly between 31,000 to 41,000 feet in the air and reach their maximum cruise height within 17 to 20 minutes of takeoff. Airlines typically maintain their cabin pressure at 7.8 to 8.65 psi during flight. Since the pressure at 8,000 feet of elevation is around 7.8 psi, airline cabin pressure roughly corresponds to the pressure at which our patient experienced his symptoms while driving in the mountains. The retinal hole discovered by another physician most likely was an incidental finding as his symptoms failed to resolve even after treatment was administered.

Although the sphenoid wall appeared to be intact on CT, the MRI findings as well as direct visualization by endoscopy during surgery on the patient suggest that the bony sinus wall was dehiscent, allowing the transmission of pressure from the sphenoid sinus cavity to the optic nerve. Typically, studies obtained to detail thin or small bony structures, such as orbital floors and temporal bones, are performed as bone algorithms, and these are then displayed as bone windows. In this particular example, had thinner 1 to 1.5 mm sections been performed as a bone algorithm, the lateral dehiscence of the sphenoid sinus may very well have been seen adjacent to the left optic tract. This relationship is very well demonstrated on the high resolution coronal MRI study (Figure 3).

Alternately, pressure may have been transmitted to the optic nerve through a very thin bony wall separating the sphenoid sinus cavity from the optic canal which protruded into the sphenoid sinus. In a study of 150 CT scans of patients without sphenoid disease, 8% were found to have protrusion of the optic canal into the sphenoid sinus.[4] In all of these cases, an extremely thin bony shell surrounded the nerve, and pneumatization of the ipsilateral anterior clinoid process was present. In the case described by Som et al,[2] it was inferred that pressure was transmitted through an intact but thinned bony sinus wall.

Other conditions known to cause visual loss as a result of sinus disease include the orbital apex syndrome which occurs in the setting of acute sphenoethmoiditis and is manifested by visual loss and ophthalmoplegia without signs of orbital pathology such as proptosis, chemosis, or lid edema.[5] Unlike the case presented previously, that condition is caused by the contiguous spread of infection into the orbital apex. In such cases, the compression and inflammation within the orbital apex result in a poor prognosis, with permanent visual loss being the usual outcome even with medical and surgical intervention.[5]

Visual loss resulting from sphenoethmoid mucoceles and, more rarely, sinus neoplasms also have been reported.[6,7] Mucoceles impinge on the optic nerve directly through a defect in the bony wall of the optic canal, either by absorption that occurs from expansion of the mucocele or erosion secondary to inflammation from infection originating in the mucocele, commonly referred to as a mucopyocele.[6] Unlike the case presented previously, visual changes caused by infection, mucoceles, or neoplasms, while capable of transience at least initially, would not be expected to be related to altitude.

Conclusion

Sphenoid sinus pneumoceles are rare but have the potential for significant morbidity, including pressure headaches, visual disturbances, and even blindness. The diagnosis may be delayed, as in the patient described who saw several specialist physicians prior to the confirmatory diagnosis and definitive therapeutic intervention. Any symptoms linked with altitude changes should raise the consideration of alternobaric related pathophysiology such as a sinus pneumocele.

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Salmonella enteritidis Infections from Shell Eggs: Outbreaks in California

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IN CALIFORNIA, BOTH the number of *Salmonella enteritidis* (SE) cases and the proportion of *Salmonella* cases due to the SE organism have increased markedly. SE cases increased from 594 in 1990 to 2,495 in 1995. Prior to 1990, less than 10% of *Salmonella* isolates were SE but since 1994 this proportion has been >25%.

Intact shell eggs can contain SE because infection of a chicken's reproductive tract can result in contamination prior to shell formation[1]; also, *Salmonella* can penetrate intact shells. Isolation of the organism from eggs is a rare event even when eggs from flocks implicated in outbreaks are tested. The prevalence of SE infection in eggs is estimated to be 1 in 10,000;[2] only 1% of eggs from infected chickens contain SE.[3] Sporadic SE infections have been associated with eating raw or undercooked eggs.[4–8] Outbreaks of SE infections have occurred primarily in the northeastern United States.[9]

We now present the first outbreak investigations in California that found a definitive association between SE disease and egg consumption and that first isolated SE in intact eggs epidemiologically linked to human disease in California. We also present the results of a survey of egg handling practices among food services, such as hospitals and restaurants, in one California county and summarize recent SE outbreaks in California.

(Mohle-Boetani JC, Werner SB, Abbott S, et al. *Salmonella enteritidis* infections from shell eggs: outbreaks in California. West J Med 1998; 169:299–301)

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Background

Between January and March 1993, three SE outbreaks were identified in noncontiguous counties in California, two in the southern and one in the northern part of the state. In Los Angeles County, 4 patients with SE infections ate at Deli A between December 26, 1992, and January 6, 1993, in the 5 days before the onset of illness; 1 person was hospitalized. In San Diego County, 11 patients with SE infections ate at Restaurant A on February 16, 1993, within 5 days before the onset of illness; 2 were hospitalized. In Santa Clara County, 22 patients with SE infections ate food from Sandwich Shop A between February 28 and March 4, 1993, within 3 days before the onset of illness; 3 were hospitalized.

Methods

Epidemiologic Studies

To identify food items associated with these outbreaks, case-control studies were conducted. Case patients had either laboratory-confirmed SE infections or illness compatible with salmonellosis; controls had no gastrointestinal symptoms during the time frame that laboratory-confirmed cases were symptomatic. Nonlaboratory-confirmed case patients and controls were meal companions of laboratory-confirmed case patients or a convenience sample of patrons who ate at the implicated restaurant during the same time frame as laboratory-confirmed case patients. In Los Angeles and San Diego, patrons were identified through credit card receipts; in Santa Clara patrons were identified by asking Sandwich Shop A customers if they purchased food during the appropriate time frame.

Environmental and traceback studies

Food handling practices were reviewed in the implicated restaurants in the week after the outbreaks were